Vol. 71, No. 3 Established 1884 August 1954

ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE
DISEASES OF INFANTS AND CHILDREN
JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

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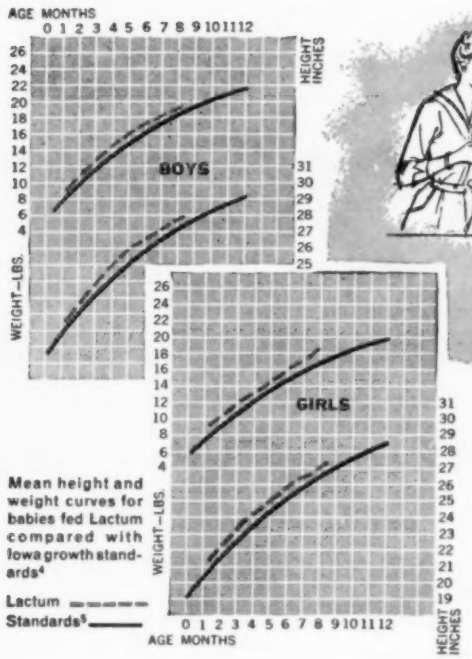

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1. Anderson, H. C., and McCarty, M.: Am. J. Med. 8.445 (April) 1950.
2. Stollerman, G. H., et al.: Am. J. Med. 15:645 (Nov.) 1953.
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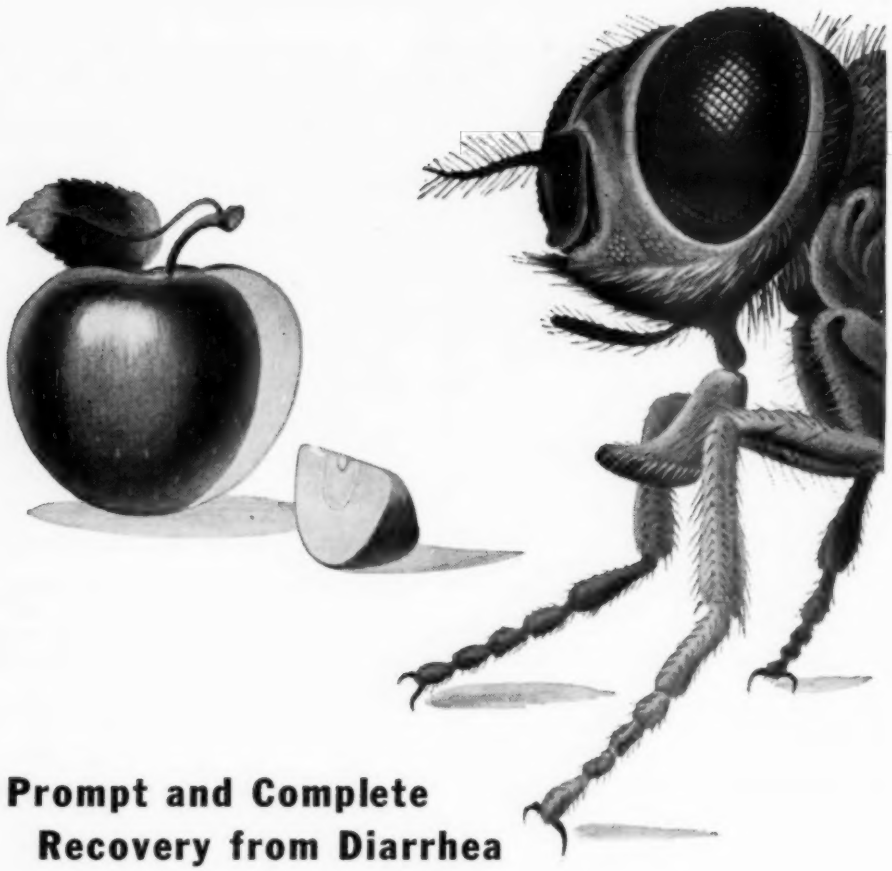

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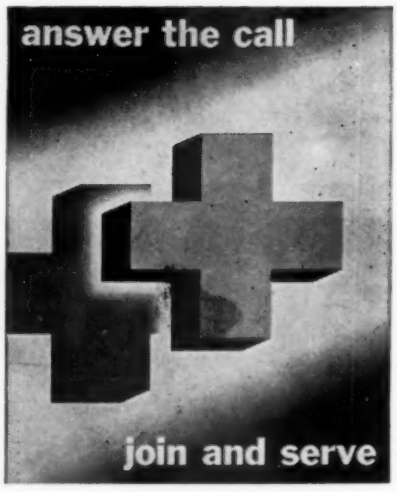

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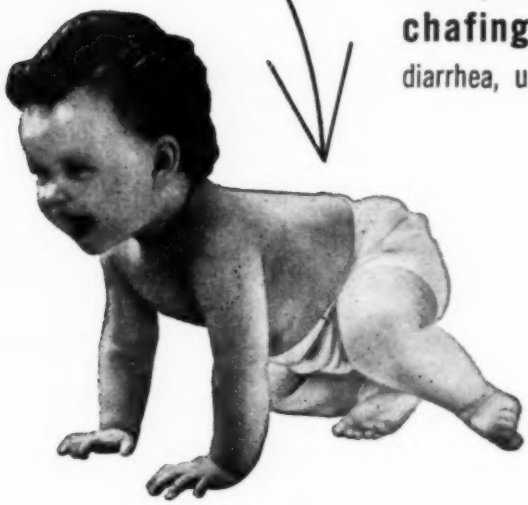

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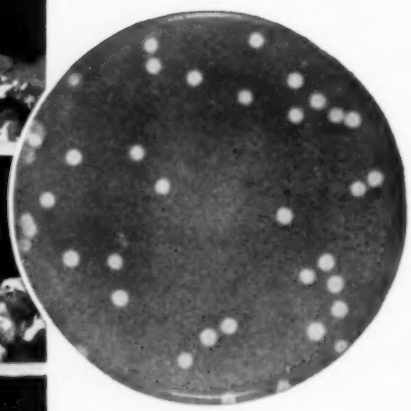

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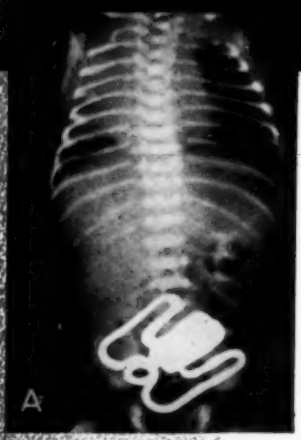

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HARMFUL SEQUELAE OF ADENOIDECTOMY IN CHILDREN WITH CHRONIC SINUSITIS

EDWARD E. BROWN, M.D., F.A.A.P.
Ashland, Ore.

This communication summarizes the ill effects of adenoidectomy in the presence of chronic sinusitis. A plea is made against routine removal of adenoids when performing tonsillectomy.

Assiduous removal of adenoids and of lymph nodules, which reform following adenoidectomy, continues to be a common practice. I obtained a history of adenoidectomy one or more times before puberty in more than half of thousands of children examined in thirteen years at a large hospital* and in ten subsequent years of private practice.

In 1932, Lemere[1] and Layton[2] independently concluded that adenoidectomy was seldom necessary and that certain disadvantages resulted from this operation. Both authors noted that adenoids rarely contained purulent material, such as is found in tonsils, and therefore did not act as foci of infection. Regarding mouth-breathing, an indication frequently used for performing adenoidectomy, both Lemere and Layton emphasized that the usual cause was nasal obstruction or nasal infection.

Four significant facts regarding tonsils, adenoids and infected sinuses are the following:

1. Tonsils and adenoids are largest in the temperate zone, being generally smaller in the tropics.[3]

2. Tonsils and adenoids are comparatively small at the end

*Study conducted at the Pediatric Chest Clinic, New York Post-Graduate Medical School and Hospital, Columbia University, New York.

of the summer,[3] becoming larger six months later in the spring of the year.

3. Tonsils are larger in children living at sea-level than at some elevation. Kaiser[3] found that they became smaller the further inland he went, presumably at higher altitudes. Holt[4] notes a decrease in the size of adenoids "in elevated regions with a dry atmosphere."

4. Chronic sinusitis is ubiquitous, particularly in the temperate zone.[5, 6] Sewall[7] is correct in stating: "The great majority of people harbor chronic sinus infection." Campbell[8] described sinusitis as the most common of all infantile diseases. Chronic suppurative sinusitis was noted in all children with frequent colds sent to our Chest Clinic, and we[9] reported its existence in 100 per cent of our rheumatic children.

How may we correlate the above four observations? Because of its ubiquity, chronic sinusitis is the common source of infection of tonsils and adenoids[10, 11] with resultant hypertrophy of these lymphoid masses.[12-14] Spontaneous improvement of sinusitis occurs under two conditions, i.e., in warm weather and in the decreased atmospheric pressures of higher altitudes. The salutary effect of high altitudes on sinusitis is observed clinically as well as experimentally in a chamber where different altitudes may be simulated.[15, 16] As the mucopurulent drainage of sinusitis abates in warm weather or during residence at increased altitudes, the bacterial assault on tonsils and adenoids likewise diminishes, with consequent decrease in size of these organs.

The source of chronic postnasal mucopurulent drainage is always sinusitis. This drainage is not reduced by tonsillectomy and adenoidectomy.[17] Dean, commenting on Kaiser's large series of cases, believes that the children had a chronic sinusitis at the time of their operation and gives this reason for continuation of the mucopurulent drip. We[18] noted the same situation in several hundred children at the New York Post-Graduate Hospital both before and after enucleation of tonsils and adenoids.

DIFFERENCES BETWEEN TONSILS AND ADENOIDS

Although histologically similar, tonsils are significantly different from adenoids in two respects. The first important difference is that tonsils may become foci of infection, whereas it is doubtful

whether adenoids ever do.[1, 19] Secondly, the adenoids are more intimately concerned with the postnasal drip of sinusitis.[1] These differences are discussed.

The tonsils may serve as a focus of infection because of their long, tortuous crypts and because of the surrounding pillars, conditions not found in adenoids. Bacteria and their toxins in tonsillar crypts may become sealed off. Toxins thus sealed under tension produce systemic effects.[20] By way of contrast, adenoids, with their shallow crypts, seldom become infected.[1, 19] Any incipient purulent content is readily expressed in deglutition. Because tonsils are surrounded by pillars, peritonsillar abscesses may result; in contrast, peri-adenoidal abscesses are nonexistent.

Tonsils and adenoids differ in their relation to rhinitis and sinusitis. The adenoids are more directly involved, being bathed in the postnasal mucopus of rhinitis and sinusitis, although to a lesser extent the tonsils are also affected by sinus secretions.[1, 11, 21-25] This mucopus causes enlargement of adenoids and, following adenoidectomy, their regrowth.[21, 23, 26-30] A granular pharynx is almost pathognomonic of chronic sinusitis.[1, 21, 26, 31]

SEQUELAE OF ADENOIDECTOMY

In evaluating the end results obtained following enucleation of tonsils and adenoids, almost all reviews, including the most recent,[32] cover the resultant health from the combined operation. It is therefore difficult to determine which of the disadvantageous sequelae, noted by Kaiser and others, are due to removal of tonsils and which to adenoids. The evidence incriminates adenoidectomy.[1, 2, 33]

As a result of removing adenoids in children with sinusitis, there is greater likelihood of involvement of the lower respiratorium.[13, 33-37] This is probably due to removal of a barrier,[34] the adenoids, against the descent of bacteria and their toxins. My experience parallels Kaiser's,[38] namely, that, following removal of tonsils and adenoids, an increased incidence may be expected in laryngitis, bronchitis, bacterial asthma and pneumonia. Bullen, in a chapter of Kaiser's book,[38] concludes that allergic symptoms were noted in "a slightly greater number" in the operated group. Walzer[39] also notes that asthma frequently follows removal of tonsils and adenoids.

Fig. 1. Boy with acute otitis media secondary to sinusitis. Tonsils and adenoids were removed one year ago. Signs of sinusitis include nasal obstruction, mouth-breathing, puffs beside nose, photophobia and cervical adenitis.

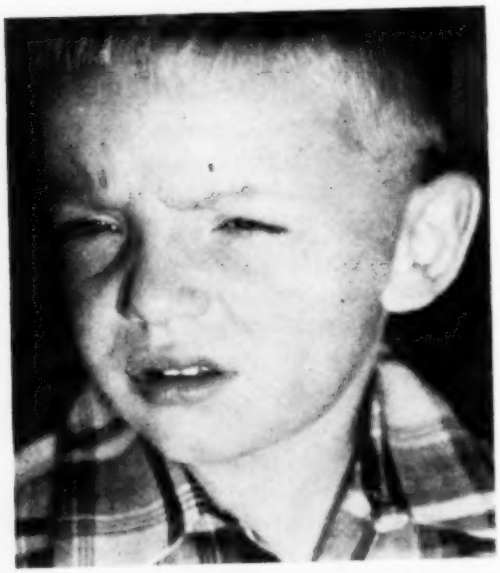

Fig. 2. Acute catarrhal otitis media, left (note cotton in ear), secondary to sinusitis. Observe photophobia, puffs beside nose and mouth-breathing secondary to sinusitis.

In our Chest Clinic, among several hundred children observed in thirteen years, although considerably more than one-half of the patients had had at least one enucleation of tonsils and adenoids, these operated children required constant observation and treatment for the various non-tuberculous infections of the lower respiratory tract. These were mainly complications of chronic suppurative sinusitis.

Mothers came to our Chest Clinic usually discouraged because much had been promised for the tonsil and adenoid operation. When colds continued or coughs returned, often with increasing frequency and severity, it was only following treatment directed to the sinuses, including a vaccine made from the sinus secretions, that these children were aided considerably.[40, 41] As already implied, these lower respiratory symptoms increased, not because of the loss of the faucial tonsils, but because of the loss of adenoids, which act as a barrier to protect the lower respiratorium from the suppurative drainage of nasal sinusitis.

To circumvent lower tract complications of ubiquitous sinusitis, for many years I have not advised adenoidectomy, except for true adenoidal obstruction. The tonsils are enucleated when diseased; only those adenoids which obstruct the passage of the finger, while the child is under anesthesia, are removed.

The *protective function of adenoids* against ear complications constitutes the final topic for discussion.

Adenoids are frequently removed to aid deafness and recurrent otitis media, a procedure of questionable value[36] (Figs. 1 and 2). It is commonly stated that overgrowth of adenoid tissue may in some instances compress the eustachian orifice. One must remember that the eustachian tube is normally closed,[42, 43] and that it opens only "by contraction of its dilator muscles and at such times any pressure differential existing between the middle ear and the atmosphere is equalized."[42] It opens during swallowing, yawning and other similar physiological acts.[42] True compression of the ostium by adenoids is unusual.[25] The Ballengers[44] state: "The adenoid does not often afford a mechanical obstruction to the patency of the tubes as it grows from the posterior and superior walls of the nasopharynx, and therefore does not involve, as a rule, the regions of the eustachian orifices on the lateral walls."

The following observations militate against the relation of

adenoids to deafness etiologically, and raise doubt as to the efficacy of adenoidectomy therapeutically. Deafness almost invariably continues following adenoidectomy (Fig. 3), and the incidence of recurrent otitis is not lessened by this procedure.[1, 45, 46] Indeed, deafness may be increased by overzealousness in removing every vestige of adenoids along with underlying mucous membrane, for trauma or laceration to the mucous cartilaginous portion of the pharyngeal ostium of the eustachian tube (torus tubarius) may produce considerable scar tissue[1, 47, 48] and prevent proper ventila-

Fig. 3. Severe bilateral deafness, chronic catarrhal otitis media, photophobia and mouth-breathing, all secondary to chronic sinusitis in a 6-year-old girl. Deafness improved shortly after treating nose.

tion. Furthermore, deafness and otitis media are apparently less common in warm equable climates, where adenoidectomy is not practiced. Kaiser[3] found no "ear troubles" in the tropics. As a corollary to this statement, in the temperate zone we commonly note that deafness improves in summer weather, only to become aggravated by the first colds[49] in the fall.

By what logic can we hope to aid deafness and recurrent otitis in assiduous radiation* and enucleation of small granules of

*Hoople[32] states: "I have seen numerous five-year follow-up audiograms of children who have been treated with radium, and in the aggregate, the hearing at the conclusion of this period showed no improvement over the pretherapeutic test."

lymphoid tissue which return following adenoidectomy, when, in tropical countries, the unremoved adenoids cause no trouble? These granules, as already emphasized, are evidence of a desperate effort on the part of the defense mechanism to fight the infection (chronic sinusitis) which produces their regrowth. Why not attack this source?

These views are shared by others. A few representative quotations follow:

McAuliffe,[50] discussing the results of treatment of deafness by tonsillectomy and adenoidectomy, states: "The catarrh which was causal remains untreated and it is no uncommon thing to see children suffering from otitis and nasopharyngeal inflammation after a thorough exenteration of lymphoid masses. . . . Where the tonsils alone have been removed, the incidence of otitis media is apparently favored."

Marantz[51] states that of the complications of sinusitis, "otitis media with its occasional termination into mastoiditis ranks foremost. This is another condition in which adenoids receive most blame and the sinuses are overlooked."

Kaiser states that "ear infections are seldom benefited to any great degree. . . . More mastoid operations are reported in tonsillectomized children than in those with tonsils." (One must equate "tonsillectomized" to include "adenoidectomized" since the operation was a combined one.)

Glover and Wilson,[46] discussing results following removal of tonsils and adenoids, note: "Possibly otitis media and mastoiditis were more frequent in the tonsillectomized."

Reger-Tuskan[52] states: "The tonsillectomized group showed a greater incidence of otitis media." (Here, again, the word "tonsillectomized" is used to include removal of adenoids.)

Lemere,[53] commenting on his practice of performing tonsillectomy without adenoidectomy, states: "very few patients . . . returned with acute suppurative otitis media, either discharging or requiring paracentesis. This at least shows that diseases of the middle ear are not more prevalent when the adenoid is left. It is possible that they are less frequent."

The role of sinusitis in otitis and deafness is most intimate.[25, 54] "Colds" produce otitis and aggravate deafness.[49, 55] The frequently recurring cold is likely to be an exacerbation of chronic sin-

usitis.[41, 56] The intimate relation of sinusitis to otitis and deafness is noted as follows:

Cullom[57] finds an incidence of 85 per cent sinusitis in patients with otitis. He quotes Campbell who, in 1932, reported an incidence of 97½ to 100 per cent sinusitis in otitis, and Fowler an 86 per cent incidence of sinusitis in diseases of the ear. Cullom comments: "To my mind this study of Dr. Fowler's constitutes one of the most important contributions that has been made to the study of deafness. Since 1920 I have had the growing conviction that nearly all forms of deafness are the result of infection and that the infection for the most part is by continuity and not blood borne."

Taylor[58] states: "Virtually all cases of otitis media are interdependent on infection of the nose or sinus."

Martin[59] writes: "Otitis media comes by way of the nasopharynx and nose; there would be much less infection of the ear, and generally much less deafness if more attention were paid to the nose."

Crooks and Signy[23] state that "normal hearing may return by antral lavage."

Lemere[1], reporting the results of 300 tonsillectomies without adenoidectomy, writes: "Nasal and sinus infections are greater factors in pathologic conditions in the ear than the mechanical obstruction of adenoids. . . . So far I have had no request for the removal of adenoids that were left undisturbed, and I have seen no indication to advise it. Colds seem less frequent; no ear troubles supervene, and nasal breathing becomes progressively more free." Five years later, after performing 500 tonsillectomies leaving the adenoid undisturbed, he[53] writes: "I have been encouraged by the reports of those who have carried out this plan of conservation of the adenoid and who have spoken or written to me."

Nasal obstruction is usually caused by chronic sinusitis.[1, 23, 60, 61] Often there is an allergic component. Other causes of nasal obstruction are edematous turbinates, polypi, foreign body, deviated septum or nasal deformity, snuffles of congenital syphilis and, extremely rarely, congenital atresia of the choanae.[62]

In chronic sinusitis intelligent cooperation on the part of parents

can usually remedy nasal obstruction and its resultant mouth-breathing. The presumed existence of adenoids and, in children previously adenoidectomized, the presumed regrowth of adenoids can be shown to be either nonexistent or innocuous when treatment is directed to the nose. Briefly, the treatment impressed on parents of affected children is the application of isotonic astringent nasal drops, the teaching of children one year of age and older to blow the nose, the avoidance of chilling and the use of the head-up position during recumbency.[63] In older children nose drops are followed by the "barber treatment,"[64] i.e., application twice daily of hot moist compresses over the face, after first applying cold cream. In infants and young children, one may substitute steam inhalations.

CONCLUSIONS

1. Adenoids should seldom be removed. Occlusion of the nasopharynx constitutes the main indication for adenoidectomy; such occlusion is uncommon.

2. Many cases of mouth-breathing are wrongly ascribed to the effects of adenoids and clear up when the nose is treated. This is particularly true when adenoids have once been removed, and in older children whose adenoids tend to atrophy.

3. Neither adenoids nor tonsils manufacture mucopurulent postnasal discharge which, when chronic, has its source in infected sinuses.

4. Adenoids act as a barrier against complications in the lower respiratory tract. Removal of adenoids increases the likelihood of laryngitis, bronchitis, bronchiectasis, bacterial asthma and pneumonia, all dependent mainly on an associated (complicating) chronic sinusitis.

5. Removal of adenoids is not often likely to aid deafness, recurring otitis media and mastoiditis. There is evidence that adenoidectomy may aggravate these conditions, since they are dependent largely on sinusitis. Treatment of sinusitis, on the other hand, is imperative.

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PUNCH BIOPSY OF LIVER IN CHILDREN. (Minerva Pediatrica, Turin, 5:265, April 30, 1953). Punch biopsy of the liver has become a valuable means of diagnosis of hepatic disease in children. The authors report their results in seven children, 1 to 14 years old. These children were given injections of vitamins C and K and barbiturates or opiates 24 hours before the intervention. After they had fasted for 12 hours, a Van-Silverman needle was inserted through a small incision into the 9th intercostal right space under local anesthesia. The procedure did not last more than eight seconds, and a 2 to 3 cm. specimen was easily obtained. The small lesion was not sutured but was protected with sterile gauze and an ice bag was placed on the area while the child was instructed to stay in bed for 12 hours. The histological specimens obtained were far superior to those the authors had obtained from other children by means of needle biopsy. The diagnosis of liver disease was confirmed in each child. There were no complications.—*Journal A.M.A.*

CONGENITAL DERMAL SINUS COMPLICATED BY MENINGITIS*

Report of Two Cases

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A congenital dermal sinus is an epithelial lined tube which extends inward from the skin and may connect the surface of the body with the central nervous system. Congenital dermal sinuses differ from the usual pilonidal sinuses in that they may occur at any location from the skull to the sacrococcygeal area. The sacrum, skull, lumbar and thoracic areas have been the most common sites reported[1].

Congenital dermal sinuses are believed to be the result of incomplete separation of the epithelial ectoderm from the neuroectoderm at the third to fifth week of fetal life[2]. Cardell and Laurance[3] reported in 1951 that there had been 34 cases recorded in the medical literature. Since that time there have been several additional reports of congenital dermal sinuses which communicated with the central nervous system, and there undoubtedly have been many unreported cases.

The signs and symptoms characteristic of this abnormality may be separated into two groups: (1) those referable to the skin lesion, and (2) those referable to central nervous system complications.

Skin. A small dimple or pinpoint opening is seen over or close to the midline of the back or scalp. The sinus opening may be covered with normal hair, if it is in the scalp. Sinuses occurring in the scalp are frequently overlooked and may require shaving of the scalp for detection. Hairs may protrude from the sinus, which may or may not be surrounded by a pigmented area or capillary hemangioma.

Central Nervous System Complications. Meningitis is the most frequent central nervous system complication. However, dermoid and epidermoid cysts may occur at any point along the course of the sinus, especially near the connection with the central nervous system. Therefore, symptoms referable to increased intra-

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cranial or intraspinal pressure may result from an expanding cyst or abscess.

Spina bifida is frequently associated with this abnormality, but in some cases the vertebrae are normal. Carefully taken roentgenograms of the skull may demonstrate a smoothly outlined, circular mid-line defect when the sinus is located in the skull. The treatment of choice is complete surgical removal of the entire sinus tract from the skin to its deepest termination.[4] This should be done as soon as the condition is recognized and when no infection exists.

The case histories of two infants with congenital dermal sinuses connecting the skin with the central nervous system will be presented. The first case is of unusual interest since the infant had meningitis on two occasions with complicating subdural serous effusions with both infections. Subdural effusions complicating bacterial meningitis in infants have been assumed to result from an altered permeability of the pia-arachnoid permitting transudation of fluid to the subdural space.[5]

CASE REPORTS

Case 1. J. J. This white male infant was born on April 4, 1953, following a normal pregnancy and labor. This was the first pregnancy for this 26-year-old mother. Birth weight was 8 pounds 3½ ounces. Physical examination at the time of birth was reported as negative, except for hypoplasia of the second, third, fourth and fifth toes of the left foot and a non-elevated capillary hemangioma, 2.5 centimeters in diameter, in the midline over the lumbosacral area of the spine. The infant's neonatal course was uneventful, and he was discharged from the Hospital on April 15, 1953, on breast feedings.

He remained healthy the first five and one-half months of life. He was given a normal infant diet with vitamin supplements. His development was considered to be within normal limits, although he showed relatively little interest in toys and his surroundings, and failed to hold his head and back up quite as well as expected for his chronological age.

On October 3, 1953, he had a sudden onset of fever (103° F., rectally) and extreme irritability. Physical examination at this time was noninforming, except for the previously noted abnormalities and moderate pharyngitis. He was treated with intramuscular

penicillin and oral Gantrisin and seemed greatly improved the following day. However, on October 5, 1953, fever and irritability were again noted. Physical examination at this time revealed, in addition to the previously noted findings, a tense anterior fontanel. The infant was admitted to Carle Memorial Hospital for further examination.

Lumbar puncture done at this time produced turbid cerebrospinal fluid under increased pressure. The fluid contained 2,950 cells per cubic millimeter with 60 per cent neutrophiles, 39 per cent lymphocytes, and 1 per cent red blood cells; the cerebrospinal fluid protein and sugar were 156 milligrams per 100 cubic centimeters and 40 milligrams per 100 cubic centimeters, respectively; no bacteria were seen on examination of the gram stained smear of cerebrospinal fluid sediment. A complete blood count revealed the hemoglobin to be 9.1 grams; erythrocyte count, 3,350,000; and leucocyte count, 12,200, with 75 per cent neutrophiles, 2 per cent stabs, 20 per cent lymphocytes, and 3 per cent monocytes. The urinalysis was negative.

A diagnosis of spinal meningitis, probably due to Hemophilus influenza, type B, was made. The infant was started on oral chloromphenical, 200 milligrams per kilogram per day; subcutaneous sodium sulfadiazine, 250 milligrams per kilogram per day; and intramuscular penicillin, 1.0 million units twice a day. Subcutaneous fluids and other supportive measures were administered. Further examination at this time revealed an opening 2.0 millimeters in diameter over the spine in the middle of the previously noted hemangioma. There was no drainage from the dimple and there was no evidence of infection. Blood sulfa levels on October 6, 8 and 12 were 16.8 milligrams per 100 cubic centimeters, 20.4 milligrams per 100 cubic centimeters, and 13.8 milligrams per 100 cubic centimeters, respectively. A marked improvement was noted on the first three days following hospitalization. However, on October 9, 1953, a rise in temperature was again observed. Oral fluids were vomited, he became quite lethargic and irritable, and he appeared critically ill. Cerebrospinal fluid was obtained again for examination. The fluid appeared slightly opaque; the total cell count was 1,712, with 1,638 erythrocytes, 50 neutrophiles and 24 lymphocytes; the spinal fluid protein and sugar were 38 milligrams per 100 cubic centimeters and 52 milligrams per 100 cubic centimeters, respectively.

Due to the infant's extremely critical condition and the marked reduction in the leucocytes in the cerebrospinal fluid, the possibility of a complicating subdural serous effusion was considered likely. Therefore, bilateral subdural punctures were done and 3.0 cubic centimeters of cherry-colored serous fluid were removed from the right subdural space. This subdural fluid was loaded with red blood cells and had 20 white blood cells per cubic millimeter. The total protein was 492 milligrams per 100 cubic centimeters. Cultures of the spinal fluid specimens previously obtained and the subdural fluid failed to grow bacteria. A striking improvement was noted in the infant's condition during the following twelve hours. Bilateral subdural aspirations were repeated the following five days. On October 10, 1.5 and 2.0 cubic centimeters of blood-tinged serous fluid were removed from the left and right subdural spaces, and on October 11, 10.0 cubic centimeters of serous fluid were aspirated from the right subdural space. No fluid was obtained on subsequent subdural aspirations.

The infant continued to improve and remained afebrile after October 9. Penicillin and chloromphenical were discontinued on October 15. The patient's developmental level was essentially as noted just prior to the onset of the illness. He was eating well, played with toys, and held his head and back up fairly well with support. He was discharged from the Hospital on October 16, 1953.

The patient gained weight well and seemed healthy the following three months. At age eight and three-fourths months he weighed 21 pounds 3 ounces and was 30½ inches long. He had been sitting without support for over three weeks, but failed to attempt to support any weight on his feet or to try to pull to a standing position.

On February 2, 1954, he suddenly developed fever, vomiting and irritability. Physical examination, eight hours after the onset, revealed only fever and the previously recorded congenital abnormalities. The anterior fontanel was depressed slightly and the neck and back were supple. A lumbar puncture was done immediately, since he had previously had meningitis and had a dimple which could represent a sinus opening over the lumbosacral spine. The cerebrospinal fluid was again turbid and contained 9,689 cells, 9,172 of which were neutrophiles. The cerebrospinal fluid protein and sugar were 106 milligrams per 100 cubic centimeters and 66

milligrams per 100 cubic centimeters, respectively. Gram stain of the fluid sediment was negative for bacteria. Complete blood count revealed a leucocytosis with a predominance of polymorphonuclears. Urinalysis was negative. Roentgenograms of the lumbar spine were negative.

The infant was again hospitalized and was started on the same therapeutic regime for meningitis that he had received three and one-half months previously. Once again he improved rapidly during the first twenty-four hours of treatment, but on February 4, 1954 he became extremely lethargic, vomited all oral fluids, and became semicomatose. All medications and fluids were administered parenterally and bilateral subdural aspirations were done. Two cubic centimeters and 4 cubic centimeters of blood-tinged serous fluid were removed from the right and left subdural spaces, respectively. Blood-tinged fluid continued to drain spontaneously from the site of aspiration on the left for the following eighteen hours. Twelve hours after the subdural aspirations were done his temperature began to fall and definite improvement was noted in his general condition. On February 7, five days after his second admission to the Hospital, subdural aspirations failed to produce fluid. On February 9, a lumbar puncture was done. Cerebrospinal fluid was clear and contained only 21 leucocytes. The cultures of cerebrospinal fluid and subdural fluid were negative for bacteria. Despite intravenous fluids, a blood transfusion, and parenterally administered antibiotics, the infant's condition once again started to deteriorate. His general condition remained poor; he was unresponsive, febrile, and had the general reactions of a three months old infant. On February 13 and 14, 10 and 12 cubic centimeters of xanthochromic fluid were removed from the right subdural space. Once again a gradual improvement followed the removal of subdural fluid. On February 15 chloromphenical was discontinued and he remained afebrile after February 16, 1954. On February 21, he sat in bed alone, played with his toys and was fairly happy. He still refused to support his weight on his feet and to make any effort to stand.

Small doses of sulfadiazine were continued the following three weeks, and on March 9, 1954, four and one-half weeks after the onset of his second illness, surgical exploration of the sinus opening over the spine was carried out. The sinus tract entered the

epidural space at the lumbosacral area and ascended craniad to where it appeared to enter the dura just superior to the fourth lumbar lamina on the right side. Complete removal of the sinus tract was accomplished. The infant had an uneventful postoperative course and was discharged from the Hospital on March 18, 1954. The patient has been followed at monthly intervals since his discharge from the Hospital and has remained healthy. His developmental level is considered to be slightly below that expected for his chronological age.

Case 2. J. W. C. This white male infant was born on December 1, 1952, following an essentially normal pregnancy and labor. The delivery was normal, and the birth weight was 7 pounds 9 ounces. The only abnormality observed on the initial examination was overriding of the parieto-occipital sutures. The infant's neonatal course was normal, and he was discharged from the Hospital on the seventh day of life on an evaporated milk formula. On the eleventh day of life he had a mild omphalitis which was treated with intramuscular penicillin and alcohol compresses. At the age of three weeks he was treated for a mild naso-pharyngitis, and at the age of five weeks he was hospitalized for seven days for treatment of bilateral bronchopneumonia. He continued to have frequent upper respiratory infections throughout the first year of life. He received an adequate infant diet with vitamin supplements. Growth and development were entirely normal.

On November 18, 1953 (age eleven and one-half months), he was hospitalized at Carle Memorial Hospital because of pallor, vomiting and listlessness. He had been ill four days and had received two injections of Distrycillin during this time. Physical examination at the time of admission to the Hospital revealed an acutely ill infant who appeared pale and mildly dehydrated. Rectal temperature was 99° F. The ears, nose and throat, heart, lungs and abdomen were normal. The anterior fontanel was closed. The neck was stiff, grade I-II. There was no back stiffness. Deep reflexes were physiological. Lumbar puncture at the time of admission to the Hospital revealed cloudy cerebrospinal fluid under apparently normal pressure. The cerebrospinal fluid contained 1,833 cells per cubic millimeter, 94 per cent neutrophiles, 4 per cent lymphocytes, and 2 per cent red blood cells. The protein and sugar were 56 milligrams per 100 cubic centimeters and 68

milligrams per 100 cubic centimeters, respectively. Cerebrospinal fluid gram stain and culture were negative. A complete blood count was reported as follows: hemoglobin, 8.4 grams; erythrocytes, 2,970,000; leucocytes, 22,200 with 42 per cent neutrophiles, 2 per cent stabs, 54 per cent lymphocytes, and 2 per cent eosinophiles. Urinalysis was negative except for sugar, 1 plus; sugar was not present in subsequent urinalyses.

Since the bacteriological type of meningitis was unknown, the infant was started on oral chloromphenical and parenteral sodium sulfadiazine and penicillin. He was given electrolytes parenterally, a blood transfusion, and other supportive measures. His hospital course was excellent, and the cerebrospinal fluid was entirely normal on November 25, 1953. All medication was discontinued on November 29, except an orally administered triple-sulfa preparation. This was discontinued two days after his discharge from the Hospital on December 2, 1953.

The infant remained well following discharge from the Hospital except for recurring mild upper respiratory infections. During an examination at the time of an upper respiratory infection, a dimple, 3.0 millimeters in diameter, was observed about one-fourth inch to the right of the midline at the upper level of the gluteal crease. It was felt that this represented a congenital dermal sinus and that it probably was the source of his previous meningitis. Therefore, on April 29, 1954, the sinus opening was explored surgically. The sinus was followed down to the dura and was completely excised. The infant's postoperative course was uneventful and he was discharged from the Hospital on May 1, 1954. His physical examination and development were normal at this time.

SUMMARY

Two cases of congenital dermal sinuses connecting the skin with the central nervous system and complicated by meningitis have been presented. The first case is of unusual interest since the infant had meningitis twice, both times complicated by subdural serous effusions.

These cases serve to illustrate the ease with which congenital dermal sinuses may be overlooked and should serve to emphasize the need for careful examination of the skin over the spine and

midline of the scalp in any infant or child who has pyogenic meningitis. Congenital dermal sinuses over the spine or scalp should be explored and completely excised surgically.

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ADRENAL CORTICAL FAILURE IN MENINGOCOCCIC SEPTICEMIA. (Lancet, London, 2:907, Oct. 31, 1953). Two patients, a 15-year-old boy and a 45-year-old man, with meningococcic septicemia and adrenal cortical failure were given specific treatment with Eucortone, a proprietary preparation of cortical extract of the suprarenal gland, and cortisone, in addition to penicillin, sulfamethazine, and sulfadiazine for treatment of the infection, and oxygen therapy for control of anoxia. Both patients survived. During the critical stage of the illness, frequent large doses of Eucortone given intravenously and of cortisone given by mouth are advised; cortisone should be given orally in an initial dose of 100 mg. followed by 25 mg. every six hours until the infection is controlled, and the patient is out of danger, the dose being reduced to 12.5 mg. every six hours for the following two days and the total daily dose being thereafter halved every second day. An initial dose of 50 cc. of Eucortone intravenously should be given and followed by another dose of 20 cc. an hour later and further doses of 10 cc. hourly until the systolic blood pressure is maintained at 80 mm. Hg. The younger patient received a total dose of 290 cc. of Eucortone parenterally within five days and 200 mg. of cortisone intramuscularly and an additional 200 mg. of cortisone orally within seven days. The older patient was given a total dose of 810 cc. of Eucortone parenterally and 437.5 mg. of cortisone orally within seven days. These doses are large, but the demands on the adrenal cortex are known to be great in the presence of severe infection. The oxygen tent seems to be superior to other methods of oxygen therapy and proved lifesaving in the younger patient.—*J.A.M.A.*

EXPERIMENTS WITH COLLODION CAPSULES FOR IMMUNIZATION AGAINST POLIOMYELITIS AND OTHER INFECTIONS*

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A previous publication[1] described cooling applied to the back in poliomyelitis cases, observations having shown the possibility of reducing spinal fluid temperatures by as much as 10° F. by this method. The purpose was two-fold: (a) the paralytic lesions are attributed chiefly to direct destruction of nerve cells by invasion of virus, and it is a general rule that infectious microorganisms grow or multiply more slowly at reduced temperatures; (b) there have been suggestions[2] of asphyxial damage of nerve cells from inflammatory swelling impeding circulation, and hypothermia is known to inhibit inflammatory reaction, diminish local metabolic needs and thus to protect the spinal cord[3] as well as other tissues against asphyxia. At the same time the method employed keeps the general body temperature normal so that production of antibodies is unimpaired, the overall idea being a retardation of local nervous damage until the systemic resistance can terminate this self-limited disease. As the obscure publication is not generally known, the following practical and theoretical features may be noted: (1) The 32 cases studied were not numerous enough to establish the benefit of a clinical treatment, and if such benefit exists it can be only minor except for its real importance in the later stages of some bulbar cases, for controlling hyperpyrexia and reducing the metabolic demands on respiration. (2) The cases sufficed to demonstrate that normal body temperature can be maintained in poliomyelitis at any stage easily and without untoward effects. This sharp distinction from the bacterial infections has theoretical interest in showing the nervous rather than toxic origin of the fever in poliomyelitis, the control being comparable with the beneficial use of hypothermia in fever due to central nervous trauma.

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The cost of approximately three thousand dollars for this research was paid by Mr. Sherman M. Fairchild and Dr. Frank K. Safford, Jr.

The writer thus became interested in further exploration which, like the temperature regulation, should be based on broad biological principles beyond the narrow limits of virology. Two denials of a requested grant of five thousand dollars caused the undertaking to be delayed over five years and to be finally carried out under extremely restricted conditions. The plan was suggested by reports on culture of microorganisms in collodion capsules outside or inside the body.[4] In brief, a considerable number of investigators have, since 1891 (Sanarelli), used this method with a wide variety of organisms, for providing favorable growth conditions, collecting toxins[5, 6] and other special studies. Surprisingly, in all this time there is no recorded instance of implantation of such sacs in animals for observing reactions on the part of the host.* Such capsules which can be graded for passage of small or large molecules or aggregates should provide ideal means for studying such reactions, and the lodgment of fully virulent organisms where they have free diffusional interchange with the body, but cannot invade, may sometimes offer advantages for immunization over the usual injections of dead or attenuated organisms.

This paper summarizes primitive observations on over three thousand mice, a smaller number of rats and a few guinea pigs and rabbits, with the following infectious agents.

VIRUSES

A radically new attempt to immunize against poliomyelitis seemed important at a time when customary vaccination methods were showing no promise. Monkeys being unobtainable, the experiments were limited to mice.

COLUMBIA SK VIRUS

The deadliness of this virus assures positive results and dispenses with statistical comparisons. Inoculations are fatal in dilutions up to several millions and, according to Jungeblut,[7] as high as 1 part in 20 million or in a billion. Apparently a mouse is doomed if a single virus particle enters the body by cerebral or any peripheral route, recovery being unknown. The time of death varies with the route and the dose. An intracerebral inoculation

*A recently discovered exception (Jungeblut[10]) will be discussed later.

commonly kills in two days or less. With large peritoneal or subcutaneous injections, paralysis and death may come within a few days, while with infinitesimal doses the mice may appear in full health for 5 or 6 weeks and then come to paralytic death either suddenly or after a few days of decline. Even with the long latency, no cure or protection by any vaccine or other means has ever been reported.

In a single preliminary experiment, a collodion capsule approximately 0.7 cm. long and 0.3 cm. thick was filled with brain tissue from a mouse that had died of the SK paralysis, and was implanted subcutaneously in a mouse's flank (Mouse A). Four weeks later 0.1 cc. of a 1/10,000 suspension of SK brain tissue was injected subcutaneously in this mouse, and the same in a control mouse. The control died with paralysis in 7 days; mouse A remained well. Five weeks later immunity was assumed to be high, due to the capsule plus the "booster" dose of living virus. Therefore, 0.1 cc. of a 1/100 suspension of SK brain was injected subcutaneously in this mouse and in a new control. The control died on the third day. Mouse A retained apparently perfect health for 32 days, then, after 3 days of decline, died with paralysis. The brain showed full typical infectivity for other mice. This first fortunate experiment was practically an epitome of the successful part of the subsequent work.

Similar capsules were therefore implanted confidently in a large series of mice, most of which successively died with paralysis during the next few weeks, their brains exhibiting the usual infectivity for other mice. Evidently the capsules, made of pharmacopoeial "flexible collodion," permitted escape of some virus particles. More than fifteen hundred mice were used in an unsuccessful attempt to make uniformly dependable capsules. Purer collodion, prepared for histological embedding, did not greatly change the results. Variations of size and wall thickness (up to 2.5 mm. thick) also gave no solution. The first capsules were made by molding on glass, then thickening by dipping in collodion solution. Later it proved more convenient to mold the collodion on druggists' gelatin capsules, either half-capsules which were later joined, or whole capsules which were later filled through a needle puncture at one end. The gelatin was at first removed with warm water, but later was left as being harmless. Results were uniform in one

peculiar respect, namely, that a capsule which had killed one mouse within any period from a few days to a few weeks invariably caused early or late death in an indefinite number of mice to which it was successively transplanted. It was expected that microscopic flaws in a capsule could be sealed by added collodion, but the baffling fact remained that a capsule which had killed one mouse continued to kill others in spite of repeated dips in collodion solution.

Poorer results were obtained with other membranes—cellophane, pliofilm, Visking sausage casing—, also failures were complete with an added thin coat of shellac or waterproof varnish followed by a final dip in collodion. There was also no success with mixing collodion with substances designed to alter permeability or inactivate virus particles passing through, such as castor oil, iodine, salicylic acid, picric acid, carbol fuchsin, gentian violet. Instead of this empiricism the scientific method of Elford,[8] using reasonably pure solvents and acetic acid, improved the results somewhat, but circumstances did not permit the necessary precision.

There were, furthermore, some indications of possible impairment of capsules in the body, in the fact that a capsule which had been carried safely by a mouse for several weeks or months and had immunized it, might prove fatal to another mouse to which it was transplanted. Though these capsules remained firm and free from any grossly perceptible leak, it is conceivable that a better product might have been obtained by spreading collodion over metal mesh, as some other investigators have done.

An obvious question of the influence of bacterial contamination was settled by preparing capsules which were filled with aseptically removed mouse brain and which were proved sterile by aerobic and anaerobic cultures before and after implantation in mice. There was no difference in the effect on the animals, and thus no evidence that bacterial action was responsible for opening up flaws in the collodion. Therefore, sterility was ignored in the great mass of the work, and smears or cultures from capsules which had remained some time in animals showed varieties of cocci or bacilli.

But though the ordinary contaminating organisms were harmless to the animals and to the virus, several instances were encountered when mice had died in hot weather and putrefactive bacteria had evidently reached their brains. Capsules from such material

rapidly killed series of mice to which they were successively transplanted, the toxemia being symptomatically distinguishable from the usual paralysis. When the same capsules were implanted subcutaneously in rats, they set up abscess formation and sloughed out. There is obvious danger in using the capsule method for any organisms producing powerful diffusible toxins.

The contents of capsules which had remained in mice for several weeks or months seemed to be as potent in producing SK paralysis as the fresh mouse brains. Evidently the virus did not die out spontaneously, and was not killed either by the accompanying bacteria or by body fluids of the immunized host. Examples of persistence in capsules containing flaws were the following: Five capsules were selected, each of which had caused paralytic death within periods of 5 days to 2 weeks in a series of 5 mice to which it had successively been transplanted. These 5 capsules were implanted subcutaneously in rats, where they remained smoothly healed for 4 months. The contents of each capsule then proved highly potent for producing SK paralysis in mice. Though the rat is absolutely immune to SK virus, earlier workers showed that its serum does not kill the virus. Also this attempt to obtain modification or attenuation with capsules failed. Poliomyelitis virus has been preserved by glycerine and refrigeration for periods up to 4 or 6 years.[9] The foregoing experiments, together with abundant survival of the virus in capsules in mice for periods up to 6 months, are unexampled for the persistence of this type of virus at body temperature, especially in the presence of bacteria. Inasmuch as autolysis destroys poliomyelitis virus in 20 to 30 days at 37° C.,[9] there is a question whether something in the exchange diffusion with the host contributes to preservation of the virus, even though it cannot multiply.* This also touches the larger question of viral metabolism.

With the available technique it was accidental whether more or fewer mice in any lot would die of paralysis or survive. There was no exception to the rule that the survivors acquired immunity. Observations concerning this immunity are the following:

*Jungeblut[10] found that poliomyelitis virus survives less than 48 hours in sacs in the monkey peritoneum, but 10 days in rabbits. Jameson and Parkinson's "Synopsis of Hygiene", 9th Ed., London, 1947, states: "The virus may persist for a hundred days in sterile water stored in the dark, and modern methods of chlorination leave it unaffected."

1. All the early implantations were made under the skin of the flank, back or belly, because the effectiveness of the subcutaneous location must be demonstrated if there was to be any practical use of the method. When the capsules are too large or are poorly placed, they gradually ulcerate out by pressure. Otherwise, they remain surrounded by a rather thick envelope of fibrous tissue. Later, in a smaller number of mice, the capsules were inserted intraperitoneally, according to the practice of previous investigators. Aside from a few deaths from peritonitis or bowel obstruction, these capsules lie harmlessly in a thin covering of omentum. No differences were noticed between the subcutaneous and peritoneal sites as regards paralytic deaths or immunity.

2. The unpredictable deaths created difficulty in learning the time of beginning of immunity, but it certainly is not evident before a period between 2 and 3 weeks after capsule implantation. Ordinarily the preference has been to wait 4 weeks or longer before the first challenge injection of virus, in order, as far a possible, to avoid confusion with the deaths from faulty capsules. Immunity is definitely demonstrable within 3 to 4 weeks. But if a mouse is given an infinitesimal subcutaneous inoculation which requires 4 to 5 weeks to cause death, and if a capsule is implanted at the same time, the mouse invariably dies.

3. It may be speculated that immunization is always due to escape of virus from the capsule, and a faster or slower rate of escape may determine whether a mouse dies of paralysis or becomes immune. This supposition is fortunately excluded by the deadliness of the SK virus, which assures that escape of any virus particles before immunity is established must positively be fatal. Therefore, it is certain that immunization is brought about by dialyzed products, and virus particles themselves are not essential to the process. The writer's attempts to prepare a collodion filtrate of SK or MEF virus as a vaccine have been thwarted by unfavorable conditions.

4. The immunity is present as long as the capsule is carried. Following its removal no instance of loss of immunity has yet been found. The longest of these observations was in a mouse which carried a subcutaneous capsule for 3 months and was found immune 4 months after its removal. This time is not long

absolutely but is a sufficient fraction of the life of a mouse to suggest a durable immunity. Immunity seems not to be strengthened by implantation of two or three capsules instead of one. It is not known whether the length of time for which a capsule is carried affects the degree or duration of immunity.

5. Perhaps because the mouse species lacks any detectable trace of natural resistance to the SK virus, the artificial immunity is limited in degree. There is no protection against intracerebral inoculation; the test mice die from infinitesimal doses and within approximately the same time as controls. The subcutaneous tolerance is not unlimited. Mice have been brought to the point of surviving an injection of 0.15 cc. of a 1/1000 suspension of mouse brain. The overwhelming dose of 0.1 cc. of 1/100 suspension was illustrated in mouse A at the opening of this section, with the remarkable prolongation of survival. The tolerance can be defined more sharply, with the inability to withstand 0.25 cc. of 1/1000 suspension. An illustration is a mouse with a subcutaneous capsule, which resisted 0.1 cc. and 0.15 cc. of 1/1000 suspension at suitable intervals. When 0.25 cc. of 1/1000 suspension was injected subcutaneously, the control died in 8 days and the test mouse in 6 weeks. The mechanism of this lengthening of latency is not explained.

6. Before and during the present work, the writer has tried vainly to protect mice by means of killed or attenuated vaccines from mouse brain. An ideal opportunity seems to be offered by the capsule-bearing mice, which can tolerate doses of fully virulent virus. The procedure is rather tedious, for it involves first the carrying of a capsule for 2 months or longer to exclude deaths due to capsule faults, then subcutaneous injection of increasing doses of SK virus 5 or 6 weeks apart to make sure that each dose is tolerated. Hopes based on these "booster" doses of living vaccine were disappointed. The failure to raise immunity above that obtained with the capsules alone appears discouraging, for the efficacy of vaccines in general against the SK virus, though not excluding possibilities for the MEF and other poliomyelitis viruses, or for other animal species.

7. The record to date, therefore, is that the capsule method affords protection against the SK virus which has not been obtained with vaccines. Elford's gradocol membranes are known to be capable

of holding back particles smaller than poliomyelitis virus while still permitting passage of chemical molecules. The dependability of such membranes has established them as a standard measure of particle size. Facilities for making accurate membranes might have avoided the great waste of work and of mice, also the consequent fears of the clinical employment of capsules.

MEF VIRUS

The mouse-adapted MEF strain was of interest as being a true poliomyelitis virus; also, being less deadly than the SK virus and infecting mice only on intracerebral inoculation, it offered a test whether the capsule method can immunize against cerebral injections, with an anticipated absence of accidental deaths due to flaws in capsules. The capsules were inserted intraperitoneally in the majority of some six hundred mice used for these experiments.

The results could not be as positive and constant as with the SK virus. With minimal doses both the test mice and the controls sometimes survive. Very large doses kill both kinds. The optimum dose may vary somewhat with age, weather and different lots of mice. Nevertheless, by using graded doses, it was possible to find instances in which the test mice survived while the controls died, to indicate that the capsule method affords a degree of protection against intracerebral inoculations of MEF virus.

Decisiveness was spoiled by a more serious complication, namely, that the expectation of freedom from deaths due to capsule flaws was mistaken, and the loss of animals from this cause was practically as high as with the SK virus. Furthermore, when the brain tissue from animals which died was inoculated into other mice, deaths resulted not only in those receiving it intracerebrally but also, days or weeks later, in those which received it subcutaneously. Septicemia was excluded by the lateness of these deaths and the paralytic symptoms. The suspicion of contamination with SK or a "wild" virus was tested by obtaining a fresh MEF supply from Dr. Jungeblut, and finding the same results with it.

Infection of animals from faulty capsules implanted intraperitoneally or subcutaneously seemed explainable only as a result of long continued seeding of peripheral tissues from such capsules. There was an obvious way of testing this hypothesis by repeated injections. Accordingly, it was observed that although the usual

single subcutaneous MEF injection is ineffectual, when rather large doses, such as 0.2 or 0.25 cc. of 1/1000 brain suspension, are injected two or three times a week for 2 or more weeks, the mice die and their brains are infective for other mice.

The poor conditions compel recognition of the possibility of error in these results. Jungeblut mentions the confusing difficulties inherent in mice as a species, because of viruses which they commonly harbor. If correct, the findings mentioned are important as an adaptation of a virus to a peripheral route, partially comparable to adaptations to organs,[10] together with an apparent heightening of virulence. Such a result may be applicable to other kinds of viruses. It may suggest repeated exposure as a factor in the epidemiology of this disease. It may particularly emphasize danger of the capsule method of immunization unless flawless capsules are positively assured. Because of the lack of serological facilities for actual proof, it was deemed best to dismiss the entire observations on MEF virus with this tentative summary, subject to check by a virologist with proper equipment.

RICKETTSIA

Experiments were performed with the Karp strain of R. tsutsugamushi. The natural scrub typhus infection of man is followed by an immune state. Vaccination with a somewhat attenuated strain plus protection by a course of chloramphenicol for about 6 weeks induces an immunity which is durable for several months or years,[11] but the method is tedious and scarcely practical for large populations, for whom the chief dependence is control of the vector mite. The size of this organism is sufficient to preclude escapes through flaws in collodion, and insertion of subcutaneous capsules may be conceived as simpler than the present vaccination.

Capsules were filled with spleen or liver tissue from mice which had died from this infection. When implanted subcutaneously in mice they sloughed out, not slowly by pressure as happened with some of the virus capsules, but rapidly in a manner denoting strong irritation. Not a single capsule could be made to remain. The intraperitoneal introduction of such capsules created signs of irritation. Peritonitis resulted sometimes, but more often the peritoneum contained clear fluid, and the capsule, instead of being smoothly enveloped by omentum, as in the experiments with

poliomyelitis, was apt to lie free in the abdominal cavity or to be attached only by a small omental tab at some point where its surface was rough. Such mice continue to live in reasonably good health. Within periods of 3 weeks to 2 months they were challenged by intraperitoneal injection of a suspension of infected spleen or liver tissue, either with capsules *in situ* or after their removal. The Rickettsia survives readily in the capsules, as shown by the infectivity of the contents after removal. This finding harmonizes with the reported maintenance of organisms of true typhus in intraperitoneal collodion sacs.[12] Normal mice die quite uniformly in 4 to 7 days according to the amount of inoculum of this tsutsugamushi strain. The mice that had received capsules survived for the same time, it being purely accidental whether the test animal or the control died first. Even with minimum doses not the slightest sign of immunization could be detected.

This experience introduced caution in hopes that, for example, capsules containing cowpox or actual smallpox virus might have advantages over the usual vaccination including less inflammatory reaction, or that protection against rabies or yellow fever might be obtainable without the neurotoxic or other risks involved with the usual vaccines. Evidently each infectious agent will require separate tests as regards both local reaction and immunizing value.

(*To be concluded in September issue*)

TRACHEOTOMY IN POLIOMYELITIS. (Tidsskrift for Den Norske Laegeforening, Oslo, *72* : 518, Aug. 15, 1952). Tracheotomy was done, in most cases during the fall of 1951, in 12 patients with poliomyelitis, all but one of whom had bulbar paralysis. Two were between the ages of 2 and 3, and the others were between 15 and 39. There was cervicothoracal paralysis in 11 cases. The fatal outcome in five of the cases is not believed to be due to the intervention, and the survival of the seven patients is ascribed to the operation. Baekkevold and Lindeman warn that the disease often develops further after tracheotomy and that these patients require the expert attention not only of the epidemiologist but of the laryngologist, internist, and pathophysiologist. Tracheotomy itself does not offer notable difficulties if the patient is in a suitable respirator. The Kifa respirator (Freiburger-Rudbech) is considered the best. It also allows unimpeded after-treatment.—*Journal A.M.A.*

THE CHILD AS A FUNCTIONAL UNIT

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The child is a socio-biological, body-mind unit. All disease, whether physical or mental, can quite logically be regarded as an interplay between the individual and his environment. In some cases the surroundings are of the utmost importance, for if the body is infected with tuberculosis, the disease will almost invariably develop. In other instances, although the pathogenic microorganism is present, the disease does not necessarily develop, as in diphtheria or typhoid. In such a particular instance, other factors will influence the result—the physical resistance of the body —whether the child is otherwise healthy, whether he is happy or whether he seeks escape from difficulty by falling ill.

The environment means not only food and shelter, but happiness and other emotional factors as well. Neurosis is a conflict with the environment, sometimes with a situation which is really difficult, sometimes because the hereditary factor has made the child unable to cope with his troubles, and sometimes because of faulty upbringing, which has conditioned the child to react in the wrong way. All disease can be looked upon as a total reaction, both bodily and mentally, to the environment.

A child functions as a whole. He responds to all situations as a total personality, not as a mind or as a body. The meaning of life is peculiarly personal and is individual in results as related to personal and social reactions. A child is in his environment and certainly his environment is in him. He reacts with what he is to what he is not—which tends to make him other than he was. He matures physically and emotionally by this process. A child is constantly active in all planes. He attempts to mould his world and he resists being moulded by it.

A child responds to stress, to accident and to disease as a body-mind unit. Whatever affects his body affects his mind and his emotions, and whatever affects his mind and his emotions certainly affects his body. Starvation, fever, thirst, accidents, bodily malformations, diabetes, heart disease give rise to disturb-

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ances in the mind as well as in the body. In the same way, anxiety, fear, jealousy, anger, worry and hatred may give rise, in time, to actual physical changes in the heart, the stomach and in other vital organs. A feeling of disgust may result in nausea and vomiting—the peristaltic movements in the intestines are reversed in direction and this is accompanied by congestion of the blood vessels supplying the intestines and stomach. With frequent repetition, the lining of the intestines and stomach undergo alteration, and ulceration may follow. These may at first be temporary, but, if stress is imposed frequently, these changes will become permanent.

Emotions and thought processes are never merely psychic, for the psyche is not something that exists somewhere in space; it is an organic agent. Neither does an organic disease remain wholly organic. It goes to the very core of the personality. Processes that are purely organic do not exist; nor do processes that are purely psychological exist as such, for the body is fundamentally a psychophysiological entity.

Mental and emotional conflicts become intelligible only in relation to personal and social development in the presence of a communal culture. Social maladjustment is the expression of the conflict of the individual with the cultural pattern as it exists objectively in his social environment.

A child's survival depends upon his fitness to survive. Fitness is adjusted to his external and internal environment and health; unfitness is maladjustment and disease. Invalidism and emotional instability find their meaning in the struggle for existence. Disease and accident are not merely matters of the body, but also of the mind. A child may respond to these in a variety of ways.

No one actually knows where physical and mental reactions begin and end. Grief and gaiety, love and hatred, jealousy and contentment, failure and success, disappointment and surprise are not mere states of feeling, but states of being. They involve the heart and lungs, the stomach, the brain, the blood vessels, the nerves. They are essentially total responses.

A painful cramp in the stomach may give rise to a fear tc eat—a fear to eat may lead to the gnawing pain of hunger. Laughter arises from a sense of the incongruous or from an organic disturbance of the power inhibiting the laughter center of the

brain. Symptoms of disease represent not only the disease process, but also the reaction of the personality afflicted by the physical condition. The reactions of fear or rage, despair, jealousy, hatred, wanting to love and fearing to love are psychosomatic and affect the power and the will to get well.

A child's utility lies in his capacity to function on physical, intellectual, emotional, social and spiritual planes. Limitations may exist upon any one plane and impair progress upon the other planes, or deficiencies in total organization may impede development upon any or all of the planes.

Mental health has been defined as personal adjustment to one's self and the world, with a maximum of personal and social effectiveness and satisfactions. The element of social responsibility is of definite importance. Social control is a requisite for mental health.

Complete isolation of human actions is impossible. Individual behavior affects social behavior; social behavior influences individual behavior. The human machine is not a thing inclusive in itself. It exists in close relationship to the industrial machine, the economic machine, the social machine. They all work in unison to fashion the world in which we live.

A child cannot exist by himself. He exists in close relation with other people and with his surroundings. A child's existence is not enclosed in the boundaries of his physical body. In his actions, feelings, intentions, thoughts, memories, the child reaches beyond himself into the surrounding world, and the world reaches into his existence.

It is quite definite that the child is a socio-biological, body-mind unit who lives in close relation with all other living creatures in the world around him. A child cannot live in himself and by himself. He cannot be isolated and insulated against his environment. His actions affect everyone and everything around him. In the same way, the world's actions constantly impinge upon him and he reacts to these influences as a body-mind unit.

DEPARTMENT OF ABSTRACTS

GUILBERT, P. W. AND BARBERO, G. J.: THE IMPORTANCE OF TRYPSIN IN INFANCY AND CHILDHOOD. PART II. CLINICAL CONSIDERATIONS. (American Journal Medical Sciences, *227*:672, June 1954).

In this review the physiology and pathological physiology of trypsin in infants and children have been described. It has been shown that pancreatic trypsin is demonstrable as early as the fourth month of fetal life and reaches an adequate level by birth. Assays for trypsin have been done by (1) duodenal intubation, (2) determination of proteolytic activitiy in the stools, (3) blood absorption studies of an ingested protein, and (4) balance studies on nitrogen intake and output as a partial reflection of the tryptic activity of the pancreas. Cystic fibrosis of the pancreas is the primary pediatric problem referable to pancreatic insufficiency and a decrease in trypsin. Transient decreases in trypsin production have been recorded in scarlet fever, mumps and acute diarrhea. No changes were found in chronic intestinal conditions and malnutrition. In the form of pancreatin, trypsin has been used for substitution principally in cystic fibrosis of the pancreas. The suggested doses have varied between 2 to 6 gm. divided into 3 daily doses with meals. Recently larger doses between 10 and 20 gm. have come into more widespread use. Pancreatin seems to show its most positive effects in marasmic children with pancreatic fibrosis. The fibrinolytic activity of trypsin has widened its role in therapy. It has been used with varying degrees of success in empyema, local debridement, bronchiectasis, atelectasis, and the pulmonary manifestations accompanying pancreatic fibrosis. The results have been encouraging enough to make it an agent worthy of careful trial and study. AUTHORS' SUMMARY.

DEMY, N.G. AND GEWANTER, A.P.: CORRELATION OF UPPER LOBE VASCULARIZATION WITH CERTAIN CONGENITAL INTRACARDIAC SHUNTS. (Radiology, 62:329, March 1954).

It should be possible for radiologists in small community hospitals to make a presumptive diagnosis of certain inoperable malformations of the heart. Early right upper lobe infiltration with

abnormal cardiac size or shape would suggest transposition of the great vessels as a first possibility and interventricular septal defect as a second. The common denominator is a large pulmonary artery. In either case, the lesion is inoperable at present and carries a fatal prognosis, death being due to congestive failure. Digitalis and other supportive measures are indicated. Atelectasis or pneumonitis is overdiagnosed and overtreated in these cases.

AUTHORS' SUMMARY.

IGALLS, T.H. AND PURSHOTTAN, N.: OXYGENATION AND RETROLENTAL FIBROPLASIA. (New England Journal of Medicine, 250:621, April 15, 1954).

Retrolental fibroplasia is the end stage of retinal vascular malformation brought on most frequently in the prematurely born infant by excesses of oxygen supply in the neonatal period of life. Other factors, such as excessive warmth and humidity, electrolyte shifts, antioxidants and enzyme derangements, probably influence onset and severity of the condition. These factors limit survival, and may determine ocular or other disease in the immature fetus. The mechanism by which retrolental fibroplasia develops in high oxygen is postulated as histotoxic anoxia brought on by the derangement of tissue enzymes at critical periods of fetal development. Clinical, experimental and epidemiologic evidence is reviewed to show that the anomaly of the retinal vascular system has been prevented by careful attention to optimal supply of oxygen. As the incubator-induced form of the disease is brought under control, it seems likely that a more intractable, sporadic form will emerge that is brought on by a concatenation of circumstances involving repeated or prolonged bouts of anoxia and some of the factors listed above. AUTHORS' SUMMARY.

POMPONIO, N.: THE TREATMENT OF PERTUSSIS WITH INTRADERMAL ACTH. (Il Lattante, 25:271, May 1954).

The author reports on the treatment of 20 cases of pertussis in children ranging in age from 6 months to 6 years. All cases showed improvement by ceasing to vomit and diminution in the severity and frequency of the coughing. The author used 1.75 to 3.5 mgr. of ACTH intradermally, depending on the age of the child. This was given daily for 9 to 13 days, depending on the response.

MICHAEL A. BRESCIA, M.D.

BOOK REVIEWS

MÉNINGITE TUBERCULEUSE & TUBERCULOSE MILIAIRE DE L'ENFANT. LEUR TRAITEMENT. By Robert Debré and H. E. Brissaud with the collaboration of J. Raynaud, S. Balsan, G. Bouveau, R. Grumbach, S. A. Kaplan, J. Lavat, J. Le Pintre, A. Lerique-Koechlin, P. Mozziconacci, M. Naveau and N. Noufflard. Paper. Pp. 632. Illustrated. Price 5,000 fr. Paris, France: Masson & Co., 1953.

This volume comprises a detailed study of 644 cases of tuberculous meningitis which were observed and treated between January 1, 1947 and October 1, 1952. In the course of their study of these cases the authors made some pertinent observations regrading the etiology, pathology, pathogenesis, cerebrospinal fluid findings, electroencephalographic studies, treatment and sequelae. The final mode of treatment for tuberculous meningitis is still not with us, and naturally some of their ideas of treatment, especially with regard to intrathecal therapy, have undoubtedly changed. The ophthalmological findings are well illustrated. This volume is of great value for anyone who is particularly interested in tuberculous meningitis.

MICHAEL A. BRESCIA, M.D.

ENDOCRINOLOGY IN CLINICAL PRACTICE. Edited by Gilbert S. Gordon, M.D., Ph.D. and H. Lisser, M.D. Cloth. Pp. 407. Illustrated. Price $10.50. Chicago: The Year Book Publishers, Inc., 1953.

This book on endocrinology is particularly aimed at the physician whose field of interest is other than endocrinology. Thus the book is most practical and very helpful. There are thirty separate authors and, although this is usually a disadvantage, it does not seem so in this volume. The authors were well-chosen and the approach has been diagnosis and therapy with a minimum of theoretical discussion. There are many subjects in this volume of interest to the pediatrician. However, of particular interest to the physician who deals with children, would be the following chapters, viz: Diabetes Mellitus in Children, by F. S. Smyth; Sexual Precocity—Types and Treatment, by H. Lisser and W. A. Reilly; and Undescended Testes, by D. R. Smith. The book is highly recommended.

MICHAEL A. BRESCIA, M.D.

THE ALLERGIC CHILD. By Harry Swartz, M.D. Cloth. Pp. 297. Price $3.95. New York: Coward-McCann, Inc., 1954.

This book was patently written to try and help the distraught mother of a child with allergy to help her try and understand the problem she is facing. The author falls far short of his goal and I'm afraid might confuse her the more and compound her fears. The first chapter "From Newborn to Human Being" is neither germane to the subject matter nor the purpose of the book and could very well have been omitted altogether. The author tends to be too dogmatic and yet indulges in self adulation and unnecessarily displays his knowledge by giving all possible names for each separate condition described, e.g., coeliac disease; Gee-Herter disease; infantile idiopathic steatorrhea; chronic intestinal indigestion. Certainly this display of erudition is not necessary in a book for the layman. Several obvious errors of fact were noted and some nebulous ideas are offered as medical truisms.

MICHAEL A. BRESCIA, M.D.

PEDIATRIC PROBLEMS IN CLINICAL PRACTICE. Edited by H. Michal-Smith, Ph.D., Cloth. Pp. 310. Price $5.50. New York: Grune & Stratton, 1954.

This volume contains 13 essays in addition to a brief foreword by Howard A. Rusk. The 13 essays cover the following subjects: The Normal Child, The Sick Child, The Emotionally Disturbed Child, The Schizophrenic Child, The Mentally Retarded Child, The Brain-Injured Child, The Cerebral Palsy Child, The Orthopedically Handicapped Child and separate essays covering the allergic, cardiac, diabetic, epileptic and tuberculous child. Each essay is written by one well-known for his interest in each particular topic. Although the information contained in the book is not new and is easily available in the many texts covering each individual subject, nevertheless in this small volume many specialists combine to point out interesting facets of their particular subject. The chapters on the Sick Child and the Cardiac Child interested this reviewer the most. MICHAEL A. BRESCIA, M.D.

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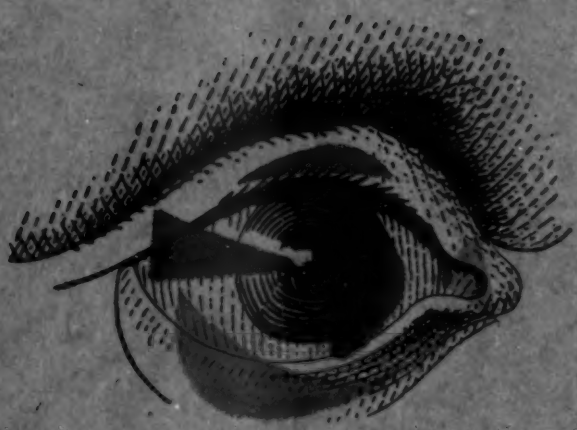

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